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NEWS 10	FEB 23	Several formats for image display and print options discontinued in USPATFULL and USPAT2
NEWS 11	FEB 23	MEDLINE now offers more precise author group fields and 2009 MeSH terms
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NEWS 13	FEB 23	Three million new patent records blast AEROSPACE into STN patent clusters
NEWS 14	FEB 25	USGENE enhanced with patent family and legal status display data from INPADOCDB
NEWS 15	MAR 06	INPADOCDB and INPAFAMDB enhanced with new display formats
NEWS 16	MAR 11	EPFULL backfile enhanced with additional full-text applications and grants
NEWS 17	MAR 11	ESBIOBASE reloaded and enhanced
NEWS 18	MAR 20	CAS databases on STN enhanced with new super role for nanomaterial substances
NEWS 19	MAR 23	CA/CAplus enhanced with more than 250,000 patent equivalents from China
NEWS 20	MAR 30	IMSPATENTS reloaded and enhanced
NEWS 21	APR 03	CAS coverage of exemplified prophetic substances enhanced
NEWS 22	APR 07	STN is raising the limits on saved answers
NEWS 23	APR 24	CA/CAplus now has more comprehensive patent assignee information
NEWS 24	APR 26	USPATFULL and USPAT2 enhanced with patent assignment/reassignment information
NEWS 25	APR 28	CAS patent authority coverage expanded
NEWS 26	APR 28	ENCOMPLIT/ENCOMPLIT2 search fields enhanced
NEWS 27	APR 28	Limits doubled for structure searching in CAS REGISTRY
NEWS 28	MAY 08	STN Express, Version 8.4, now available
NEWS 29	MAY 11	STN on the Web enhanced

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 NEWS 30  MAY 11  BEILSTEIN substance information now available on
                  STN Easy
 NEWS 31  MAY 14  DGENE, PCTGEN and USGENE enhanced with increased
                  limits for exact sequence match searches and
                  introduction of free HIT display format
 NEWS 32  MAY 15  INPADOCDB and INPAFAMDB enhanced with Chinese legal
                  status data

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=> E "OOPC"/CN 25
E1            1     OOLONGTHEANIN-3'-O-GALLATE/CN
E2            1     OOMYCIN A/CN
E3            0 --> OOPC/CN
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E4            1     OOPG 1000/CN
E5            1     OOPG 1002/CN
E6            1     OOPLASM SPECIFIC PROTEIN (MUS MUSCULUS STRAIN NIH/SWISS GENE
OP1)/CN
E7            1     OOPODIN/CN
E8            1     OOPODIN, 11,13-DIDEHYDRO-/CN
E9            1     OOPORPHYRIN/CN
E10           1     OORA SUBUNIT OF 2-OXOGLUTARATE:ACCEPTOR OXIDOREDUCTASE
(CAMPYLOBACTER JEJUNI STRAIN NCTC 11168 GENE OORA)/CN
E11           1     OORA SUBUNIT OF 2-OXOGLUTARATE:ACCEPTOR OXIDOREDUCTASE
(HELICOBACTER HEPATICUS STRAIN ATCC51449 GENE OORA)/CN
E12           1     OORAKKU APO 101/CN
E13           1     OORAKKU APO 101-HITALOID 3083-70B-MDI COPOLYMER/CN
E14           1     OORAKKU APO 101-HITALOID 3083-70B-MILLIONATE MR 200 COPOLYMER/CN
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E19           1     OORC SUBUNIT OF 2-OXOGLUTARATE:ACCEPTOR OXIDOREDUCTASE
(HELICOBACTER HEPATICUS STRAIN ATCC51449 GENE OORC)/CN
E20           1     OORD SUBUNIT OF 2-OXOGLUTARATE:ACCEPTOR OXIDOREDUCTASE
(CAMPYLOBACTER JEJUNI STRAIN NCTC 11168 GENE OORD)/CN
E21           1     OORD SUBUNIT OF 2-OXOGLUTARATE:ACCEPTOR OXIDOREDUCTASE
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E23           1     OOSPGLYCOL/CN
E24           1     OOSPOALDEHYDE/CN
E25           1     OOSPOALDEHYDE, (2,4-DINITROPHENYL)HYDRAZONE/CN

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E1            1     OLEYLONITRILE/CN
E2            1     OLEYLOXYETHYL CIDOFOVIR/CN
E3            0 --> OLEYLOXYETHYLPHOSPHOCHOLINE/CN
E4            1     OLEYLOXYPROPYL-N,N-DIMETHYLAMINE/CN
E5            1     OLEYLPALMITAMIDE/CN
E6            1     OLEYLPHENOL/CN
E7            1     OLEYLPHOSPHORYLETHANOLAMINE/CN
E8            1     OLEYLPROPYLENEDIAMINE/CN
E9            1     OLEYLSARCOSINE N-HEPTADECYL-1,3-PROPANEDIAMINE SALT/CN
E10           1     OLEYLSARCOSINE SODIUM SALT/CN
E11           1     OLEYLSHOGAOL/CN
E12           1     OLEYLSTEARYLAMINE/CN
E13           1     OLEYLSUCCINIC ACID/CN
E14           1     OLEYLSUCCINIC ANHYDRIDE/CN
E15           1     OLEYLTRIMETHYLAMMONIUM BROMIDE/CN
E16           1     OLEYLTRIMETHYLAMMONIUM CHLORIDE/CN
E17           1     OLEYLTRIMETHYLENEDIAMINE/CN
E18           1     OLEYLTRIOCTADECYLAMMONIUM BROMIDE/CN
E19           1     OLF-1/EBF TRANSCRIPTION FACTOR (CAENORHABDITIS ELEGANS STRAIN N2
GENE UNC-3)/CN
E20           1     OLF-1/EBF-LIKE-1(8) TRANSCRIPTION FACTOR (MOUSE STRAIN CD-1
8-AMINO ACID INSERT ISOFORM)/CN
E21           1     OLF-1/EBF-LIKE-2(9L) TRANSCRIPTION FACTOR (MOUSE STRAIN CD-1
LONG ISOFORM 9L)/CN
E22           1     OLF-1/EBF-LIKE-2(OS) TRANSCRIPTION FACTOR (MOUSE STRAIN CD-1
SHORT ISOFORM 03)/CN
E23           1     OLF-1/EBF-LIKE-3 TRANSCRIPTION FACTOR (MOUSE STRAIN CD-1)/CN
E24           1     OLFACTOMEDIN (HUMAN CLONE DE10316701-SEQID-623 GENE OLFM1
ISOFORM 1)/CN
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E2           1     OLEYLONITRILE/CN
E3           0 --> OLEYLOXYETHYL/CN
E4           1     OLEYLOXYETHYL CIDOFOVIR/CN
E5           1     OLEYLOXYPROPYL-N,N-DIMETHYLAMINE/CN
E6           1     OLEYLPALMITAMIDE/CN
E7           1     OLEYLPHENOL/CN
E8           1     OLEYLPHOSPHORYLETHANOLAMINE/CN
E9           1     OLEYLPROPYLENEDIAMINE/CN
E10          1     OLEYLSARCOSINE N-HEPTADECYL-1,3-PROPANEDIAMINE SALT/CN
E11          1     OLEYLSARCOSINE SODIUM SALT/CN
E12          1     OLEYLSHOGAOL/CN
E13          1     OLEYLSTEARYLAMINE/CN
E14          1     OLEYLSUCCINIC ACID/CN
E15          1     OLEYLSUCCINIC ANHYDRIDE/CN
E16          1     OLEYLTRIMETHYLAMMONIUM BROMIDE/CN
E17          1     OLEYLTRIMETHYLAMMONIUM CHLORIDE/CN
E18          1     OLEYLTRIMETHYLENEDIAMINE/CN
E19          1     OLEYLTRIOCTADECYLAMMONIUM BROMIDE/CN
E20          1     OLF-1/EBF TRANSCRIPTION FACTOR (CAENORHABDITIS ELEGANS STRAIN N2
GENE UNC-3)/CN
E21          1     OLF-1/EBF-LIKE-1(8) TRANSCRIPTION FACTOR (MOUSE STRAIN CD-1
8-AMINO ACID INSERT ISOFORM)/CN
E22          1     OLF-1/EBF-LIKE-2(9L) TRANSCRIPTION FACTOR (MOUSE STRAIN CD-1
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SHORT ISOFORM 03)/CN
E24          1     OLF-1/EBF-LIKE-3 TRANSCRIPTION FACTOR (MOUSE STRAIN CD-1)/CN
E25          1     OLFACTOMEDIN (HUMAN CLONE DE10316701-SEQID-623 GENE OLFM1
ISOFORM 1)/CN

=> S 96720-06-8/RN
L1           1 96720-06-8/RN

=> DIS L1 1 SQIDE

L1   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2009 ACS on STN
RN   96720-06-8  REGISTRY
CN   3,5,8-Trioxa-4-phosphahexacos-17-en-1-aminium, 4-hydroxy-N,N,N-trimethyl-,
     inner salt, 4-oxide  (CA INDEX NAME)
MF   C25 H52 N O5 P
LC   STN Files:   BEILSTEIN*, CA, CAPLUS, CASREACT, CHEMCATS, CSCHEM, MEDLINE,
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DT.CA  CAplus document type:  Journal
RL.NP  Roles from non-patents:  PREP (Preparation)
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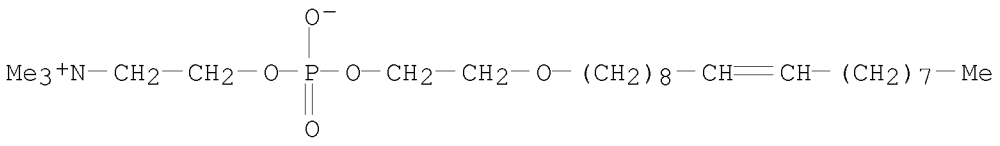

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=> s l1 or oleyloxyethylphosphocholine
             1 L1
             3 OLEYLOXYETHYLPHOSPHOCHOLINE
L2           4 L1 OR OLEYLOXYETHYLPHOSPHOCHOLINE

=> d l2 1-4 ibib abs

L2   ANSWER 1 OF 4  CAPLUS  COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER:        2004:829238  CAPLUS
DOCUMENT NUMBER:         141:329077
TITLE:                   Interactions of 12-lipoxygenase with phospholipase A2
                         isoforms following platelet activation through the
                         glycoprotein VI collagen receptor
AUTHOR(S):               Coffey, Marcus J.; Coles, Barbara; Locke, Matthew;
                         Bermudez-Fajardo, Alexandra; Williams, P. Claire;
                         Jarvis, Gavin E.; O'Donnell, Valerie B.
CORPORATE SOURCE:        Department of Medical Biochemistry and Immunology,
                         Wales College of Medicine, Cardiff University,
                         Cardiff, CF14 4XN, UK
SOURCE:                  FEBS Lett. (2004), 576(1-2), 165-168
                         CODEN: FEBLAL; ISSN: 0014-5793
PUBLISHER:               Elsevier B.V.
DOCUMENT TYPE:           Journal
LANGUAGE:                English
AB   Recent studies implicate the collagen receptor, glycoprotein VI (GPVI) in
     activation of platelet 12-lipoxygenase (p12-LOX).  Herein, we show that
     GPVI-stimulated 12-hydro(peroxy)eicosatetraenoic acid (H(P)ETE) synthesis
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is inhibited by palmityl trifluromethyl ketone or oleyloxyethylphosphocholine, but not bromoenol lactone, implicating secretory and cytosolic, but not calcium-independent phospholipase A2 (PLA2) isoforms. Also, following GPVI activation, 12-LOX co-immunoppts. with both cytosolic and secretory PLA2 (sPLA2). Finally, venoms containing sPLA2 acutely activate p12-LOX in a dose-dependent manner. This study shows that platelet 12-H(P)ETE generation utilizes arachidonate substrate from both c- and sPLA2 and that 12-LOX functionally assocs. with both PLA2 isoforms.

REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2003:112769 CAPLUS

DOCUMENT NUMBER: 139:346941

TITLE: Enzymatic activity and inhibition of the neurotoxic complex vipoxin from the venom of Vipera ammodytes meridionalis

AUTHOR(S): Noetzel, Corinna; Chandra, Vikas; Perbandt, Markus; Rajashankar, Kanagalaghatta; Singh, Tej; Aleksiev, Boris; Kalkura, Narayana; Genov, Nicolay; Betzel, Christian

CORPORATE SOURCE: Institute of Medical Biochemistry and Molecular Biology, University Hospital Eppendorf, Hamburg, 22603, Germany

SOURCE: Zeitschrift fuer Naturforschung, C: Journal of Biosciences (2002), 57(11/12), 1078-1083
CODEN: ZNCBDA; ISSN: 0939-5075

PUBLISHER: Verlag der Zeitschrift fuer Naturforschung

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Vipoxin from the venom of Vipera ammodytes meridionalis is an unique neurotoxic complex between a toxic phospholipase A2 and a highly homologous non-toxic protein inhibitor. It is an example of evolution of a catalytic and toxic function into inhibitory and non-toxic one. The activity of the V. ammodytes meridionalis toxin is 1.7 times higher than that of the closely related (92% sequence identity) neurotoxic complex RV4/RV7 from the venom of Vipera russelli formosensis. The enhanced enzymic activity of vipoxin is attributed to limited structural changes, in particular to the substitutions G54R and Q78K in the PLA2 subunit of the complex and to the T54R substitution in the inhibitor. Oleyloxyethylphosphocholine, aristolochic acid and vitamin E suppressed the enzymic activity of vipoxin and its isolated PLA2 subunit. These compds. influence inflammatory processes in which PLA2 is implicated. The peptide Lys-Ala-Ile-Tyr-Ser, which is an integral part of the PLA2 components of the two neurotoxic complexes from V. ammodytes meridionalis and V. russelli formosensis (sequence 70-74) activated vipoxin increasing its PLA2 activity by 23%. This is in contrast to the inhibitory effect of the resp. pentapeptides with 70-74 sequences on other group II PLA2s. Surprisingly, the same peptide inhibited 46% of the V. russelli formosensis PLA2 activity. The limited changes in the structure of the two highly homologous neurotoxins lead to considerable differences in their interaction with native peptides.

REFERENCE COUNT: 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2000:735786 CAPLUS

DOCUMENT NUMBER: 133:345041

TITLE: Investigation into the involvement of phospholipases A2 and MAP kinases in modulation of AA release and cell growth in A549 cells

AUTHOR(S): Choudhury, Qamrul G.; Mckay, Diane T.; Flower, Roderick J.; Croxtall, Jamie D.
CORPORATE SOURCE: Department of Biochemical Pharmacology, The William Harvey Research Institute, St. Bartholomew's and the Royal London School of Medicine and Dentistry (Queen Mary and Westfield College, London, EC1M 6BQ, UK
SOURCE: British Journal of Pharmacology (2000), 131(2), 255-265
CODEN: BJPCBM; ISSN: 0007-1188
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The authors have investigated the contribution of specific PLA2s to eicosanoid release from A549 cells by using specific inhibitors of secretory PLA2 (ONO-RS-82 and oleyloxyethylphosphocholine), cytosolic PLA2 (AACOCF3 and MAFP) and calcium-independent PLA2 (HELSS, MAFP and PACOCF3). Similarly, by using specific inhibitors of p38 MAPK (SB 203580), ERK1/2 MAPK (Apigenin) and MEK1/2 (PD 98059) the authors have further evaluated potential pathways of AA release in this cell line. ONO-RS-82 and oleyloxyethylphosphocholine had no significant effect on EGF or IL-1β stimulated 3H-AA or PGE2 release or cell proliferation. AACOCF3, HELSS, MAFP and PACOCF3 significantly inhibited both EGF and IL-1β stimulated 3H-AA and PGE2 release as well as cell proliferation. Apigenin and PD 98509 significantly inhibited both EGF and IL-1β stimulated 3H-AA and PGE2 release and cell proliferation, whereas, SB 203580 had no significant effect on EGF or IL-1β stimulated 3H-AA release, or cell proliferation but significantly suppressed EGF or IL-1β stimulated PGE2 release. These results confirm that the liberation of AA release, generation of PGE2 and cell proliferation is mediated largely through the actions of cPLA2 whereas, sPLA2 plays no significant role. The authors now also report a hitherto unsuspected contribution of iPLA2 to this process and demonstrate that the stimulating action of EGF and IL-1β in AA release and cell proliferation is mediated in part via a MEK and ERK-dependent pathway (but not through p38MAPK). The authors therefore propose that selective inhibitors of MEK and MAPK pathways may be useful in controlling AA release, eicosanoid production and cell proliferation.
REFERENCE COUNT: 65 THERE ARE 65 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1985:406131 CAPLUS
DOCUMENT NUMBER: 103:6131
ORIGINAL REFERENCE NO.: 103:1103a,1106a
TITLE: A new efficient and versatile synthesis of alkyl phosphorylcholines
AUTHOR(S): Magolda, R. L.; Johnson, P. R.
CORPORATE SOURCE: Cent. Res. Dev. Dep., E. I. du Pont de Nemours and Co., Wilmington, DE, 19898, USA
SOURCE: Tetrahedron Letters (1985), 26(9), 1167-70
CODEN: TELEAY; ISSN: 0040-4039
DOCUMENT TYPE: Journal
LANGUAGE: English
OTHER SOURCE(S): CASREACT 103:6131
AB Phosphorylcholines ROP(O)(O-)OCH2CH2N+Me3 [R = Me(CH2)n, Me(CH2)7CH:CH(CH2)8, Me(CH2)mS(CH2)3, Me(CH2)7CH:CH(CH2)8S(CH2)3, Me(CH2)mOCH2CH2, Me(CH2)7CH:CH(CH2)8OCH2CH2; m = 15,17; r = 5,7,11,17] were prepared in 35-50% overall yield by treating ROH with POCl3, followed by ethylene glycol and treating the resulting cyclic phosphates with Me3N.

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You have entered a file name of duplicates to keep that is not
referenced by any of the L#s specified for this DUPLICATE command.
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'1998' NOT A VALID FIELD CODE
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'1998' NOT A VALID FIELD CODE
L4           1 L3 AND (PD<1998 OR PRD<1998)

=> d l4 ibib abs

L4   ANSWER 1 OF 1     MEDLINE on STN
ACCESSION NUMBER:   1991183640     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 1901255
TITLE:              Inhibitors of cytochrome P-450 attenuate the myogenic
                    response of dog renal arcuate arteries.
AUTHOR:             Kauser K; Clark J E; Masters B S; Ortiz de Montellano P R;
                    Ma Y H; Harder D R; Roman R J
CORPORATE SOURCE:   Department of Physiology, Medical College of Wisconsin,
                    Milwaukee 53226.
CONTRACT NUMBER:    HL-29587 (United States NHLBI NIH HHS)
                    HL-33833 (United States NHLBI NIH HHS)
                    HL-36279 (United States NHLBI NIH HHS)
                    +
SOURCE:             Circulation research, (1991 Apr) Vol. 68, No. 4,
                    pp. 1154-63.
                    Journal code: 0047103. ISSN: 0009-7330.
PUB. COUNTRY:       United States
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
                    (RESEARCH SUPPORT, NON-U.S. GOV'T)
                    (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        199105
ENTRY DATE:         Entered STN: 26 May 1991
                    Last Updated on STN: 3 Feb 1997
                    Entered Medline: 8 May 1991
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AB The role of cytochrome P-450 in the myogenic response of isolated, perfused renal arcuate arteries of dogs to elevations in transmural pressure was examined. The phospholipase A2 inhibitor oleyloxyethylphosphorylcholine (1 and 10 microM) inhibited the greater than threefold increase in active wall tension in these arteries after an elevation in perfusion pressure from 80 to 160 mm Hg. Inhibition of cyclooxygenase activity with indomethacin (1 or 10 microM) had no effect on this response. The cytochrome P-450 inhibitors ketoconazole (10 and 100 microM) and beta-diethyl-aminoethyldiphenylpropylacetate (SKF 525A, 10 and 100 microM) also inhibited the myogenic response. At a pressure of 160 mm Hg, SKF 525A (10 microM) and ketoconazole (100 microM) reduced active wall tension in renal arteries by approximately 70%. Partial inhibition of the myogenic response was obtained after perfusion of the vessels with mechanism-based inhibitors of P-450, 1-aminobenzotriazole (75 microM) and 12-hydroxy-16-heptadecynoic acid (20 microM). The thromboxane receptor antagonist SQ 29,548 (1 or 10 microM) had no effect on the pressure-induced increase in active wall tension in renal arteries. Arachidonic acid (50 microM) constricted isolated perfused renal arteries and potentiated the myogenic response in the presence of indomethacin. This response was completely reversed by ketoconazole (100 microM) or SKF 525A (100 microM). Microsomes (1 mg/ml) prepared from small renal arteries (200-500 microns) and incubated with [1-14C]arachidonic acid (0.5 mu Ci, 50 microM) produced a metabolite that coeluted with 20-hydroxyeicosatetraenoic acid (20-HETE) during reversed-phase high-performance liquid chromatography. The formation of this product was inhibited by both ketoconazole and SKF 525A at concentrations of 10 and 100 microM. These results are consistent with the involvement of the vasoconstrictor 20-HETE and other cytochrome P-450 metabolites of endogenous fatty acids in the myogenic response.

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E "OLEYLOXYETHYLPHOSPHOCHOLINE"/CN 25
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L1 1 S 96720-06-8/RN

FILE 'CAPLUS' ENTERED AT 15:23:18 ON 27 MAY 2009
L2 4 S L1 OR OLEYLOXYETHYLPHOSPHOCHOLINE

FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 15:24:38 ON 27 MAY 2009
L3 16 S L1 OR OLEYLOXYETHYLPHOSPHOCHOLINE
L4 1 S L3 AND (PD<1998 OR PRD<1998)

=> file uspatfull

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	3.91	22.86

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-3.28

FILE 'USPATFULL' ENTERED AT 15:26:39 ON 27 MAY 2009

FILE COVERS 1971 TO PATENT PUBLICATION DATE: 26 May 2009 (20090526/PD)
FILE LAST UPDATED: 26 May 2009 (20090526/ED)

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HIGHEST GRANTED PATENT NUMBER: US7540032
HIGHEST APPLICATION PUBLICATION NUMBER: US20090133177
CA INDEXING IS CURRENT THROUGH 26 May 2009 (20090526/UPCA)
ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 26 May 2009 (20090526/PD)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Feb 2009
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Feb 2009

USPATFULL now includes complete International Patent Classification (IPC)
reclassification data for the third quarter of 2008.

=> s l1 or oleyloxyethylphosphocholine
             0 L1
             0 OLEYLOXYETHYLPHOSPHOCHOLINE
L5           0 L1 OR OLEYLOXYETHYLPHOSPHOCHOLINE

=> d his

     (FILE 'HOME' ENTERED AT 15:21:12 ON 27 MAY 2009)

     FILE 'REGISTRY' ENTERED AT 15:21:33 ON 27 MAY 2009
                E "OOPC"/CN 25
                E "OLEYLOXYETHYLPHOSPHOCHOLINE"/CN 25
                E "OLEYLOXYETHYL"/CN 25
L1            1 S 96720-06-8/RN

     FILE 'CAPLUS' ENTERED AT 15:23:18 ON 27 MAY 2009
L2            4 S L1 OR OLEYLOXYETHYLPHOSPHOCHOLINE

     FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 15:24:38 ON 27 MAY 2009
L3           16 S L1 OR OLEYLOXYETHYLPHOSPHOCHOLINE
L4            1 S L3 AND (PD<1998 OR PRD<1998)

     FILE 'USPATFULL' ENTERED AT 15:26:39 ON 27 MAY 2009
L5            0 S L1 OR OLEYLOXYETHYLPHOSPHOCHOLINE

=>

---Logging off of STN---



=>
Executing the logoff script...


=> LOG Y

COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                                 4.61      27.47

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)      SINCE FILE      TOTAL
                                                   ENTRY    SESSION
CA SUBSCRIBER PRICE                                 0.00      -3.28

STN INTERNATIONAL LOGOFF AT 15:28:12 ON 27 MAY 2009
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